IN THE SPECIFICATION:

Please substitute the following amended paragraph for the original paragraph on page 15, lines 8-25:

FIGURES ~~3a-3d~~ 3a-3j show graphs which illustrate that stable expression of CKR5 confers susceptibility to HIV entry that can be inhibited by anti-CD4 mAb or chemokines. Candidate receptors were introduced into CD4-positive and CD4-negative cell lines. Figures 3a-3c illustrates the infection of NIH3T3.CD4 cells (murine fibroblast) expressing different chemokine receptors or fusion-GFP. Fusion-GFP is a fusion protein in which Green Fluorescent Protein (GFP) has been attached to the C-terminus of fusion. In the lower panels, (Figures 3b-3c), β - chemokine and anti-CDO4 blocking are expressed as a percentage of luciferase activity in the presence of blocking agent as compared to untreated controls. Figures ~~3b~~ 3d-3g shows the chemokine induction of Ca^{2+} signaling in 3T3.CD4-CKR stable transfectants. Comparison of cytoplasmic Ca^{2+} levels in 3T3 cells expressing recombinant C-C chemokine receptors-1, -3, -5 (CKR-1, CKR-3, CKR-5), and the orphan receptor fusion after challenge with various chemokines as listed. Chemokines were added through an injection port at approximately 20 seconds (the sharp spike in each record) to a final concentration of 100 nM. The rise in intracellular calcium is represented by the rapid increase in relative fluorescence intensity. Figure ~~3c~~ 3h shows the *i*nfection of HOS.CD4 cells (human osteosarcoma). Figure ~~3d~~ 3i-3j shows the *i*nfection of Hela.CD4 (human carcinoma); US28 is a -chemokine receptor encoded by human cytomegalovirus. Duffy antigen is a promiscuous chemokine receptor expressed primarily on erythroid cells.

Please substitute the following amended paragraph for the original paragraph on page 16, lines 18-33:

FIGURE 6 is a gel showing that CC-CKR-5 is expressed in T cells and monocyte/macrophages. Total RNA was prepared from the indicated cell-types using Triazol reagent (Gibco/BRL), treated with RNase-free DNase (Boehringer-Mannheim) and used in reverse-transcriptase-PCR

reactions. First strand cDNA was primed with oligo-dT using Superscript reverse transcriptase as per manufacturer's direction (Gibco/BRL) and products were amplified with primers hybridizing to the 5' and 3' untranslated regions of CC-CKR-5 (upstream CTCGGATCCGGTGGAACAAGATGGATTAT (SEQ ID NO: 1); downstream CTCGTCGACATGTGCACAACTCTGACTG (SEQ ID NO: 2)) or to glyceraldehyde-3-phosphate dehydrogenase using a Taq/Pwo polymerase mixture (Boehringer Mannheim). To control for the presence of genomic DNA, control cDNA reactions in which reverse transcriptase was omitted were prepared in parallel. These were uniformly negative. To test the linearity of amplification, a ten-fold dilution series (lanes 1-5) starting at 1 pg of pcCKR5 plasmid DNA was amplified under conditions identical to those above. In lane 6, no DNA was added. Monocytes were prepared by overnight adherence to plastic. T cells were prepared from the monocyte-depleted preparation by adherence to anti-CD2-coated beads (Dynal).

Please substitute the following amended paragraph for the original paragraph starting on page 48, lines 28-34, continuing on to page 49, lines 1-13:

Murine cells transfected with human CD4 are resistant to infection with all tested strains of HIV. To determine whether chemokine receptors could confer susceptibility to infection, the different receptor genes are stably introduced into murine 3T3.CD4 cells. Cells expressing CC-CKR-1 (CCR1), CC-CKR-2B (CCR2B), CC-CKR-3 (CCR3), CC-CKR-4 (CCR4), Duffy, or ~~fusion~~ fusin (CXCR4) are all resistant to infection with HIV-luciferase pseudotyped with macrophage-tropic Envs, but are infected with virus bearing amphotropic Env (Fig. ~~3a~~ 3a-3c). Expression of CC-CKR-5 permitted infection with the macrophage-tropic pseudotypes, but these cells are resistant to infection mediated by HXB2 Env (Fig. ~~3a~~ 3a-3c). Only ~~fusion~~ fusin-expressing 3T3.CD4 cells are permissive for infection with this T-tropic virus (Fig. ~~3a~~ 3a-3c). The chemokine receptors are expressed on the surface of the 3T3.CD4 cells, as assessed by mobilization of intracellular free Ca^{++} in response to the appropriate chemokines (Fig. ~~3b~~ 3d-3g). Cells expressing CC-CKR-5 (CCR5) responded to RANTES, MIP-1α and MIP-1β, consistent with known β-chemokine reactivities. Infection of the 3T3.CD4 cells expressing CC-CKR-5 with macrophage-tropic virus is blocked by a mixture of the three chemokines that efficiently

activate this receptor as well as by anti-CD4 antibody (Fig. ~~3a~~ 3a-3c). Infection of the fusion-expressing cells with T-tropic virus is also blocked by anti-CD4, but is completely refractory to treatment with chemokines. Thus, these results suggest that only CC-CKR-5 mediates entry of macrophage-tropic Envs, that T-tropic envelope glycoproteins do not use this co-receptor for entry, and that β-chemokines block entry of the macrophage-tropic virus by specifically binding to this receptor.

Please substitute the following amended paragraph for the original paragraph on page 49, lines 15-23:

Stable expression of CC-CKR-5, but not of the other β-chemokine receptors, in human HOS.CD4, HeLa.CD4, and U87MG.CD4 cells also conferred upon these cells susceptibility to infection with macrophage-tropic HIV-1 (Fig. ~~3c, 3d~~ 3h, 3i-3j and data not shown). As observed in the transient transfections, stable co-expression of both CC-CKR-5 and CD4 is required for viral entry into the HeLa cells (Fig. ~~3d~~ 3i-3j). Infection of these cells with macrophage-tropic virus is reduced by 70-80% upon treatment with a mixture of chemokines (Fig. ~~3d~~ 3i-3j). High levels of β-chemokines failed to inhibit infection of HOS.CD4 cells. In general, inhibition with β-chemokines is consistently less efficient in the non-lymphoid cells expressing CD4 and CC-CKR-5 than in the PM1 cells.